purpose of this study is to evaluate glucose utilization in the isolated rat heart at baseline and with insulin stimulation using acetyl-FDG, which is taken up independently of transport. It is hydrolized and phosphorylated within the cells similarly to FDG. Hearts from fasted, male Sprague-Dawley rats were excised and perfused according to Langendorff with Krebs-Hensleit bicarbonate buffer containing Acetyl-FDG and 10 mM glucose at constant pressure of 60 mmHg. After baseline measurements, hearts were perfused with insulin (100 nM) added to perfusate. Radioactivity was monitored using BGO detector interfaced with a computer. Time-activity curves were decay corrected and normalized to radioactivity in perfusate. Slopes were calculated from fitted linear curves as an index of rate of tracer accumulation (ml/g/min). Baseline accumulation of acetyl-FDG (0.155 ± 0.009) was approximately 7 times faster than that of FDG (0.024 ± 0.005) in the same model. After addition of insulin, there was a clearance of acetyl-FDG (−0.029 ± 0.001) in contrast to results with FDG, which showed an approximately 6-fold increase in uptake compared to baseline (0.113 ± 0.057). Approximately 10 minutes after perfusion with insulin, however, acetyl-FDG was taken up again at a reduced rate (0.023 ± 0.006, 15% of baseline).

Thus, the faster accumulation of acetyl-FDG compared to FDG provide further evidence that transport is rate-limiting for glucose utilization at baseline in the isolated rat heart. The initial clearance of acetyl-FDG after insulin suggest that the increase in glucose transport stimulated by insulin increased cellular glucose concentration, which competes with acetyl-FDG for hexokinase activity, causes a net back-diffusion of the tracer. After approximately 10 minutes, glucose transport again appears to be rate-limiting; hence, the positive myocardial accumulation of acetyl-FDG. The presented dual tracer technique may provide new insights into the roles of transport and phosphorylation in the regulation of exogenous glucose utilization.

1003 Left Ventricular Function and Remodeling

Tuesday, March 18, 1997, Noon–2:00 p.m.
Anaheim Convention Center, Hall E
Presentation Hour: 1:00 p.m.–2:00 p.m.

1003-110 Can Left Ventricular Diameters be Used to Assess Left Ventricular Remodeling?

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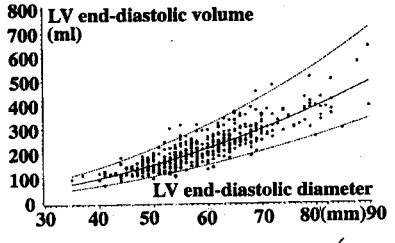
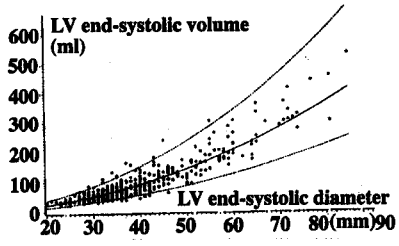
Left ventricular diameters (LV) are easy to measure and commonly used as substitutes for volumetric analysis to evaluate LV remodeling caused by LV overload or dysfunction. However, their impact on outcome is disputed, suggesting that they may not adequately assess LV remodeling. To determine the validity of LV diameters in the assessment of LV remodeling, LV diameters by M-mode and volumes by biplane Simpson's rule were prospectively and simultaneously measured in 463 patients (289 men, age 62 ± 15); 46 normal subjects, 53 with aortic regurgitation, 281 with mitral regurgitation and 83 with LV dysfunction. The LV ejection fraction (EF) and wall stress were calculated using the M-mode and 2D volumes. The correlation between diameters and volumes was good both at end-systole ($r = 0.91$, $p < 0.001$) and at end-diastole ($r = 0.86$, $p < 0.001$). However, the relation was exponential with increasing 95% C.I (see figures). The calculated EF and wall stress using LV diameters and using LV volumes correlated linearly with a limited range of error (respectively, $r = 0.95$, SEE = 5%, $p < 0.001$ and $r = 0.95$, SEE = 10%, $p < 0.001$).





We conclude that for the assessment of LV remodeling M-mode diameters 1) allow acceptable estimation of EF and wall stress and 2) correlate significantly to the LV volumes, but 3) are hindered by a wide range of error for the assessment of LV size, especially for enlarged ventricles, suggesting that LV volume measurement should be the preferred method of echocardiographic assessment of LV remodeling.

1003-111 The Dependence of the E wave on Pulmonary Venous Flow: Mathematical Solution with In Vivo Verification

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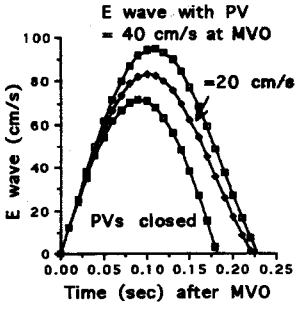
The E wave has become part of our assessment of LV diastolic function. However, it is load-dependent and varies with left atrial pressure (LAP). This pressure is supported by pulmonary venous (PV) inflow and its momentum at mitral valve opening (MVO), which may vary *independent* of intrinsic LV diastolic properties and initial diastolic LAP; for example, PV momentum at MVO is near zero in atrial fibrillation. We studied this effect in two ways: (1) Mathematically, by combining an elastance model for pressure with the unsteady Bernoulli equation into a system of differential equations to derive an analytical solution for the E wave related to PV, LA, and LV diastolic compliance and relaxation time; LA pressure and PV velocity at MVO; as well as PV and mitral inertance and area. (2) In 9 open-chest dogs under right-heart bypass to measure the E wave in the beat before and after computer controlled PV occlusion at MVO. *Results:* The mathematical simulation showed that with all other factors being equal, including initial LAP, as the initial PV velocity decreased from 40 cm/s to zero, the E wave peak velocity and deceleration time decreased to 70% of their initial values. Under comparable conditions in vivo, the same magnitude of change was observed (peak E = 70.4 ± 15.8 vs. 50.8 ± 12.1 cm/s, $p < 0.05$).



Conclusions: The initial PV flow momentum is an important additional determinant of the E-wave because it supports atrial pressure. This momentum may vary independent of intrinsic LV diastolic properties and initial LAP. Therefore the initial velocity of PV flow should be considered in physiologic assessments of diastolic function based on the E wave.

1003-112 Decrease of Mitral Regurgitation with Heart Failure Treatment is Due to a Reduction in the Regurgitant Orifice

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Although treatment of congestive heart failure is associated with a decrease in the severity of mitral regurgitation, the mechanisms for this reduction are incompletely understood. To explore mechanisms for this decrease, we performed echocardiography before and after therapy to decrease filling pressures in 12 patients with heart failure. In addition to hemodynamic variables, echocardiographic variables were measured blindly. Mitral regurgitant flow was determined from color m-mode proximal isovelocity surface areas and the effective regurgitant orifice area was calculated as: Regurgitant flow integral/regurgitant velocity integral. *Results:* Tailored therapy resulted in a decrease in systolic blood pressure (104 ± 10 mmHg to 92 ± 11 mmHg, $p = 0.005$) and pulmonary wedge pressure (30 ± 7 mmHg to 21 ± 5 mmHg, $p < 0.001$). Cardiac index increased from 1.90 ± 0.47 to 2.38 ± 0.44 L/min/m^2, ($p = 0.03$). Mitral regurgitant flow decreased from 44.4 ± 32.6 ml to 11.6 ± 9.8 ml ($p = 0.003$). Although the effective regurgitant orifice area decreased with therapy (0.38 ± 0.33 cm^2 to 0.11 ± 0.01 cm^2, $p = 0.01$), this was not due to any change in the peak Doppler-derived ventriculo-atrial systolic pressure gradient (64.4 ± 17.8 to 63.1 ± 14.4 mmHg, p = NS). *Conclusion:* Heart fail-